Psoriasis: from the genetics to the therapy

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Psoriasis is a multifactorial, chronic inflammatory skin disease characterized by increased proliferation of keratinocytes, activation of immune cells and susceptibility to metabolic syndrome. Genetic predisposition and environmental factors are both important in disease etiology. Several genome-wide association studies have been carried out and until now 36 susceptibility loci have been identified. Hyperproliferation of the keratinocytes in the psoriatic plaques is triggered by infiltrating T-lymphocytes at the dermal–epidermal junction. Autoimmune basis for chronic inflammation is supposed, although no consistent autoantigen has been found. The keratinocytes of the uninvolved psoriatic epidermis are inherently oversensitive to proliferative signals, and this elevated sensitivity plays a crucial role in the development of psoriatic lesions. Thus, resident skin cells and infiltrating immune cells cooperate in the formation of psoriatic lesions, but the exact molecular mechanisms that regulate the interactions between these cells are still far from understood. In the present overview our data on the altered response of the clinically uninvoved skin of psoriatic patients will be presented. In addition, using systems biology approach we could find novel important targets, that were previously not yet associated with psoriasis. Furthermore, analysis of chemical-protein interaction networks suggested many promising drug candidates for the treatment of the disease.